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ACCELERATION INDUCED LOSS OF CONSCIOUSNESS - A REVIEW OF 500 EPISODES

Dr. James E. Whinnery, M.D., Ph.D.
Air Vehicle and Crew Systems Technology Department (Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

Angela M. Whinnery, B.S.N.
72nd Aeromedical Evacuation Squadron
United States Air Force Reserve
McGuire AFB, NJ 08641

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Unconsciousness resulting from exposure to increased levels of head-to-foot ($+G_z$) acceleration stress (501 unconsciousness episodes) on a human centrifuge in asymptomatic, healthy subjects was investigated. A method for quantitatively measuring the kinetics of the unconsciousness and associated phenomenon was developed. In addition, a theoretical framework for describing the central nervous system (CNS) alteration resulting from acute reduction of blood flow was formulated to allow a method for defining unconsciousness phenomenon.

The length of unconsciousness and the associated incapacitation was found to be dependent on the magnitude of the CNS insult resulting from reduced blood flow. The magnitude of the insult was determined by the onset and offset rate of the $+G_z$-stress and the length of time at increased $+G_z$. The incapacitation resulting from $+G_z$-stress included 11.9s of absolute incapacitation (unconsciousness) and 16s of relative incapacitation (confusion/disorientation) for 28s of total incapacitation (period of time for lack of purposeful movement). Myoclonic convulsions were observed in approximately 70% of the unconsciousness episodes. The convulsions lasted 4s and occurred following the return of CNS blood flow. The convulsions occurred after 8s of unconsciousness and ended coincident with the return of consciousness. They occurred when the CNS insult was of greater magnitude. Memorable dreams occurred and were considered to occur during the terminal portion of the convulsion period. The dreams occurred with exposures having longer unconsciousness. The length of unconsciousness and incapacitation were affected by the wearing of an anti-G suit, with both being

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22a NAME OF RESPONSIBLE INDIVIDUAL: Dr. James E. Whinnery

22b TELEPHONE *(Include Area Code)*: 215-441-1563

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reduced if the suit was worn. Performance of an anti-G straining maneuver resulted in an increased length of incapacitation by allowing the subject to get to higher levels of $+G_z$-stress and to sustain a greater amount of acceleration exposure.

The results of this 11 year study of human unconsciousness provides a quantitative kinetic description of the phenomenon in healthy humans completely documented on videotape. These results should be of interest to neuro-psychophysiologists investigating unconsciousness, convulsive activity, and dream phenomenon. They also provide the basis for future research aimed at solving $+G_z$-induced loss of consciousness problems in fighter aircraft aviation.

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INTRODUCTION

Although much interest and effort has been devoted toward understanding unconsciousness, very little actual human research has been accomplished in investigating the actual kinetics of loss of consciousness (LOC) and the associated phenomena. Qualitative descriptions of unconsciousness induced by various techniques have provided a detailed understanding of the various psychophysiologic events that are related to LOC (1). Much of this research was performed prior to the routine ability to record the entire LOC episode. Detailed and accurate establishment of the time course of LOC and associated events is difficult without a full video recording of the episode. In addition, specific research groups had different positions of interest for investigation of LOC. The majority of the research has been associated with various medical aspects of LOC, in which the time course of acute recovery was of relatively little applied importance.

In aviation and space medicine, any reason for sudden incapacitation is of extreme concern because of the critical nature of the environment in which the personnel (aircrew) perform duties. Any alteration in consciousness, even for a few seconds, may be a threat to life, materiel, and mission accomplishment. Prevention of LOC, of any etiology, and minimization of the length of time of recovery, should LOC occur, is a dual goal of aerospace medicine and physiology. A detailed kinetic description of LOC is of vital importance to achieve these goals.

Aircrew flying high performance fighter aircraft are exposed to severe environmental stresses. A unique stress encountered in fighter aircraft combat flight is head-to-foot ($+G_z$) acceleration force. The physiologic result of $+G_z$-stress is a resulting displacement of blood away from the central nervous system (CNS) toward pooling areas in the abdomen and lower extremities. The acute reduction of blood flow to the CNS results in an ever-present risk for $+G_z$-induced loss of consciousness (G-LOC), as long

as the heart remains lower than the CNS within the $+G_z$-field. An accurate qualitative description of G-LOC and the associated psychophysiologic events has been previously given (2,3). Based on 11 years of compiling G-LOC episodes on videotape it is now possible to quantitatively describe the kinetics of G-LOC. Since all G-LOC episodes occurred in healthy, asymptomatic men, the results provide detailed information on the normal response of the CNS to acute reduction of blood flow. This information should enhance the understanding of the basic mechanisms associated with unconsciousness and subsequent recovery.

METHODS

In 1977 a centrifuge data repository was established to document the physiologic response, tolerance, and symptoms of exposure to $+G_z$-stress (4). As a part of this data repository, a videotape library of all episodes of G-LOC was archived for future evaluation of G-LOC phenomena. During the period from 1977 through 1987, over 500 episodes of G-LOC were archived into the videotape library. This study represents a review of 501 episodes of G-LOC taken from the videotape library of the centrifuge repository. Although increasing sophistication has been employed in the techniques used for monitoring G-LOC over the 11 year period, the centrifuge exposures that produced the unconsciousness episodes in themselves, remained essentially consistent over the entire period.

The subjects who experienced G-LOC consisted of experimental volunteer subjects, students in various aerospace medical disciplines, and aircrewmen. All subjects included in the study were asymptomatic and had successfully completed a U.S. Air Force flying class II physical examination or the equivalent. No subjects with known hemodynamically significant cardiac rate or rhythm disturbances induced during $+G_z$-stress were included in the study. Although an occasional subject suffered more than one G-LOC, for the most part, the 501 G-LOC episodes were from all different individuals (476 subjects and

501 G-LOC episodes. With little exception the G-LOC episodes were from on-going research and training being performed on the centrifuge. The G-LOC episodes were therefore unexpected and represent a wide range of exposure conditions.

The methods utilized in characterizing G-LOC have been described (5,6). The G-LOC events were timed using a stopwatch and with a digital clock reading (in minutes and seconds) which existed on the videotape recording. A continuous digital display of the instantaneous $+G_z$-level on the videotape was used to record the $+G_z$ related data. All measurements were made independently by the two investigators. In all cases where the measurements differed by more than 1s, the measurements were rechecked until agreement varied by no more than 1s. The onset of unconsciousness and return of consciousness were determined subjectively from videotape review. The return of purposeful movement following G-LOG was measured using an aircraft master caution light (either continuous or flashing at 0.5 Hertz) and an auditory tone (2900 Hertz, sound level 80dB). Both visual and auditory warning devices were initiated immediately upon recognition of the onset of G-LOC and subsequently extinguished by the subjects as soon as possible upon recovery from G-LOC. All subjects were thoroughly briefed on these procedures prior to $+G_z$-exposure, including the methods to manually disenable the warning devices.

A measurements profile was developed, based on the experimental exposure conditions and the symptoms related to G-LOC. This measurement profile is shown schematically in Figure 1 with the definition of each parameter provided in Table 1. Based on these experimental measurements, the important aspects of G-LOC are available or can be calculated. The G-LOC parameters, both measured and calculated, fall into three categories: $+G_z$-exposure description, incapacitation description, and myoclonic convulsion description. The ($+G_z$) acceleration exposure description is shown in Figure 2 with the parameters defined in Table 2. The incapacitation description is shown in Figure 3 with the parameters

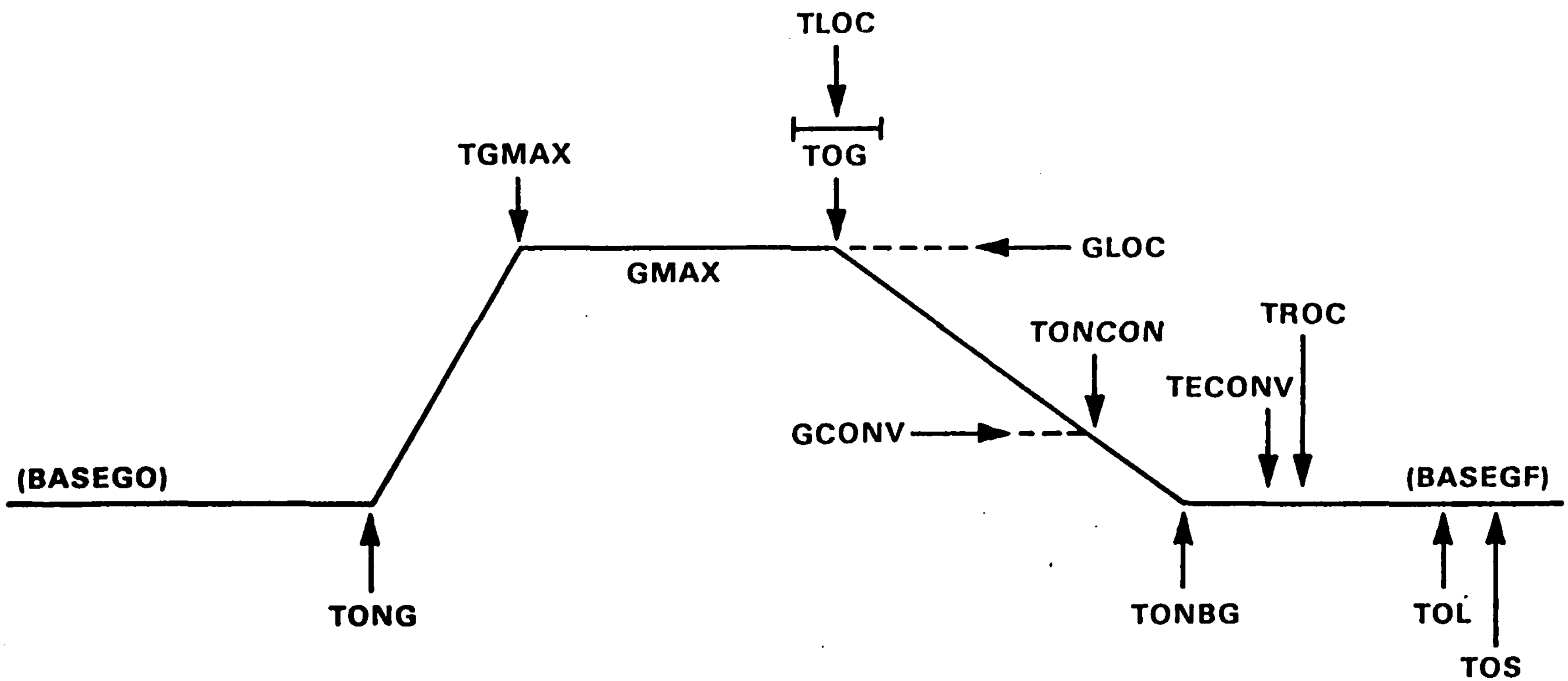

FIGURE 1. Experimental $+G_z$ and Time Measurements.

Table 1. G-LOC Experimental Time Measurements
and Exposure Description.

PARAMETER	MEASUREMENT	DEFINITION
BASEGO	$+G_Z$-units	Base $+G_Z$ at onset of the exposure
TONG	Clock time (min:sec)	Time for onset of $+G_Z$-stress
TGMAX	Clock time	Time for onset on maximum $+G_Z$
GMAX	$+G_Z$-units	Maximum $+G_Z$-level attained
TOG	Clock time	Time at offset of $+G_Z$-stress
TLOC	Clock time	Time of onset of unconsciousness
GLOC	$+G_Z$-units	$+G_Z$-level at onset of unconsciousness
TONCON	Clock time	Time of onset of convulsions
GCONV	$+G_Z$-units	$+G_Z$-level at onset of convulsions
TONBG	Clock time	Time of reaching final base $+G_Z$
TECONV	Clock time	Time when convulsions stop
TROC	Clock time	Time when consciousness returns
BASEGF	$+G_Z$-units	Base $+G_Z$ at completion of exposure
TOL	Clock time	Time for extinguishing warning light
TOS	Clock time	Time for extinguishing warning tone

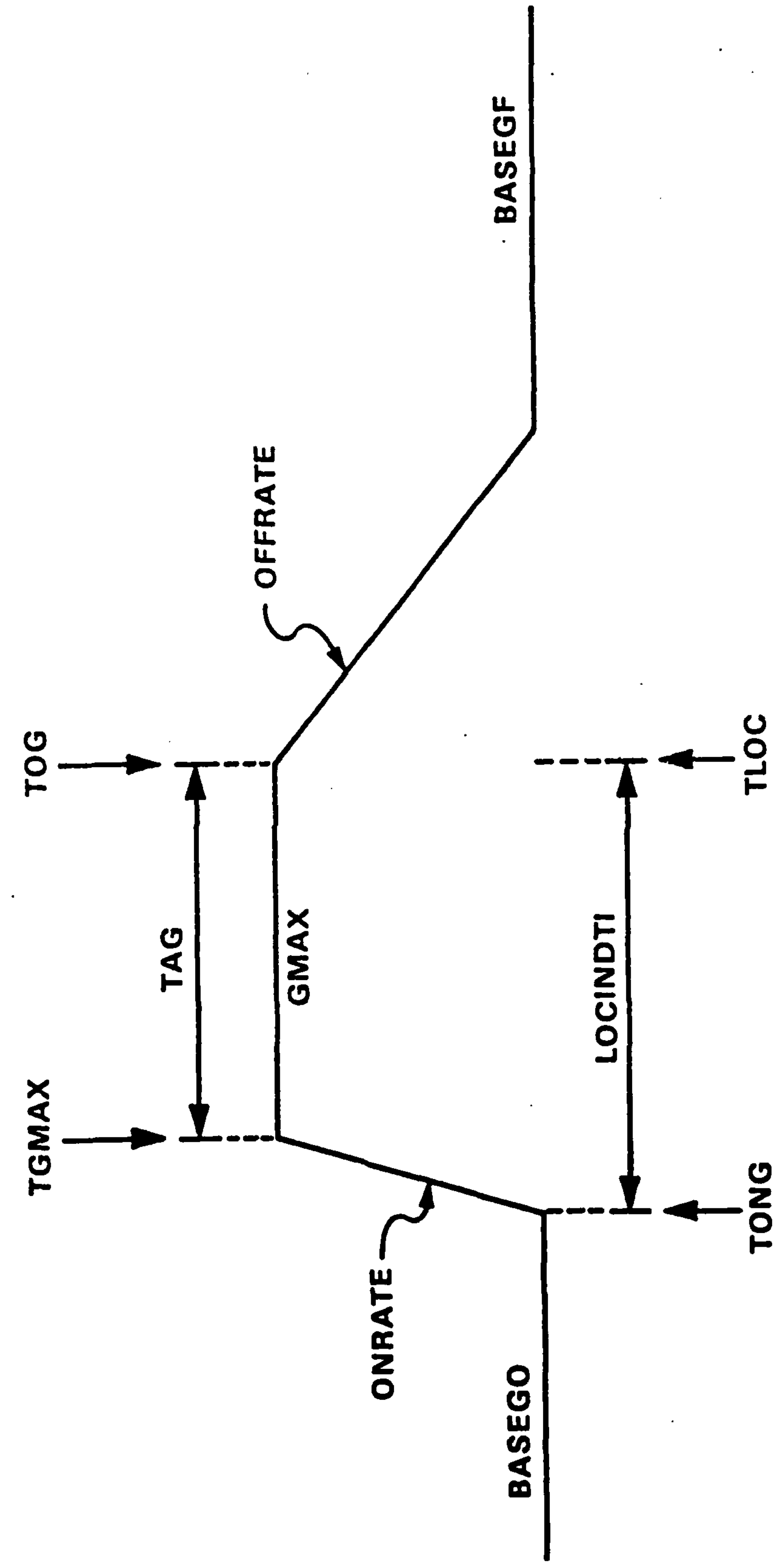

FIGURE 2. Acceleration Exposure Description.

Table 2. Acceleration Exposure Data.

PARAMETER	UNITS	DESCRIPTION
BASEGO	G	Base $+G_Z$ at onset of the exposure
ONRATE	G/s	Average $+G_Z$-onset rate of the exposure ONRATE = (GMAX-GBASEO)/(TGMAX-TONG)
GMAX	G	Maximum $+G_Z$-level of the exposure
TAG	s	Time spent at maximum G TAG = TOG-TGMAX
TOTGS	G • s	Total area under the G-time curve (See Table V for calculation)
GLOC	G	$+G_Z$-level at onset of unconsciousness
LOCINDTI	s	Length of time to induce unconsciousness LOCINDTI = TLOC-TONG
OFFRATE	G/s	Average $+G_Z$-offset rate of the exposure OFFRATE=(GMAX-GBASEF)/(TONBG-TOG)

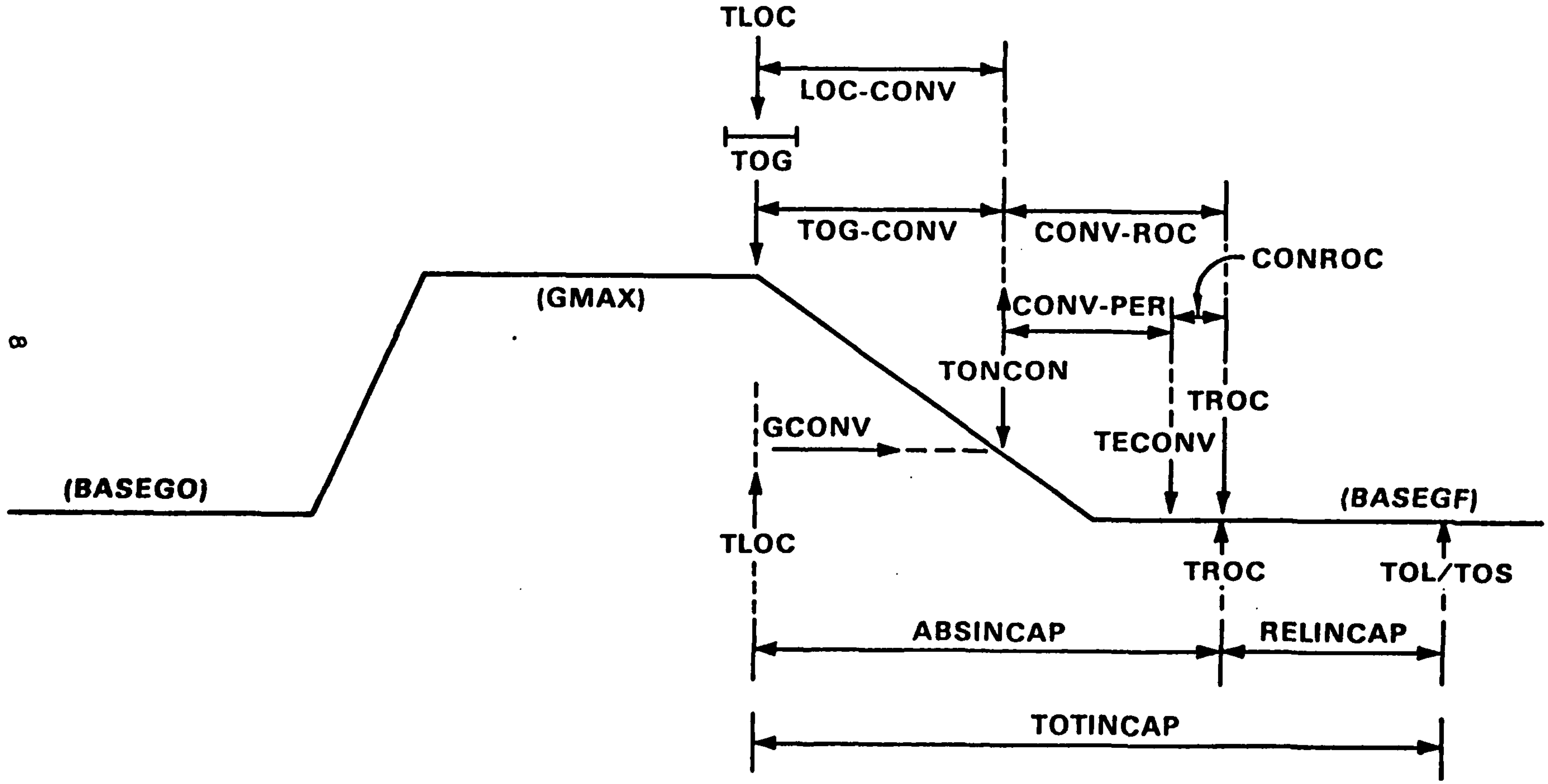

FIGURE 3. Incapacitation and Convulsion Description.

defined in Table 3. The myoclonic convulsion description is also shown in Figure 3 with the parameters defined in Table 4. The acceleration exposure, as a function of time, which induced G-LOC is described in Figure 4 and defined in Table 5.

The onset (and offset) data represent the calculated values based on the average rate of $+G_z$ onset between the initial $+G_z$-level at the beginning of the exposure and the maximum $+G_z$-level of the exposure (similar assumption made for the average offset rate). The data therefore are only relative and may not reflect the instantaneous maximum $+G_z$ onset rates achieved on the rapid onset exposures.

Other exposure variables were also recorded. Included was whether the subject was relaxed or performed a straining maneuver during the exposure. These straining maneuvers, which included a 3-5s rapid Valsalva-like respiratory maneuver concurrent with tensing the skeletal musculature, are performed to enhance tolerance to $+G_z$-stress (7). Wear or absence of an inflated anti-G suit and whether or not the subject had a memorable dream were also recorded. Collection of dream events was not initiated in the earlier years of G-LOC recording and information was therefore limited in number. With respect to the utilization of visual and auditory warning devices used to measure incapacitation, the presence or absence of external interaction with the subject was documented. External interaction was considered present if voice communication of any kind was initiated with the subject prior to his spontaneous disengagement of either warning device.

All subjects were exposed to $+G_z$-stress in a sitting position. The seat configuration was upright with either 15° or 30° tilt-back from vertical. The subjects were held in this position, throughout the $+G_z$ exposure (and G-LOC episode) and subsequent recovery, with a lap-shoulder harness.

Table 3. Incapacitation Data.

PARAMETERS	UNITS	DESCRIPTION
ABSINCAP	s	Absolute incapacitation period (unconsciousness) ABSINCAP=TROC-TLOC
RELINCAP-L	s	Relative incapacitation period(confusion/ disorientation) (as measured to extinguishing the warning light) RELINCAP-L = TOL-TROC
RELINCAP-S	s	Relative incapacitation period (as measured to extinguishing the warning tone) RELINCAP-S = TOS-TROC
TOTINCAP-L	s	Total incapacitation period (as measured to extinguishing the warning light) TOTINCAP-L = TOL-TLOC
TOTINCAP-S	s	Total incapacitation period (as measured to extinguishing the warning tone TOTINCAP-S = TOS-TLOC

Table 4. Myoclonic Convulsion Description.

PARAMETERS	UNITS	DESCRIPTION
GCONV	G	$+G_z$-level at onset of convulsions
LOC-CONV	s	Time from loss of consciousness to onset of convulsions (convulsion free period) LOC-CONV=TONCON-TLOC
TOG-CONV	s	Time from offset of $+G_z$-stress to onset of convulsions TOG-CONV = TONCON-TOG
CONV-PER	s	Length of time convulsions lasted CONV-PER = TECONV-TONCON
CONV-ROC	s	Time from onset of convulsions to the return of consciousness (convulsion prone period) CONV-ROC = TROC-TONCON
CONROC	s	Difference in time from the end of convulsions to return of consciousness CONROC = TROC-TONCON

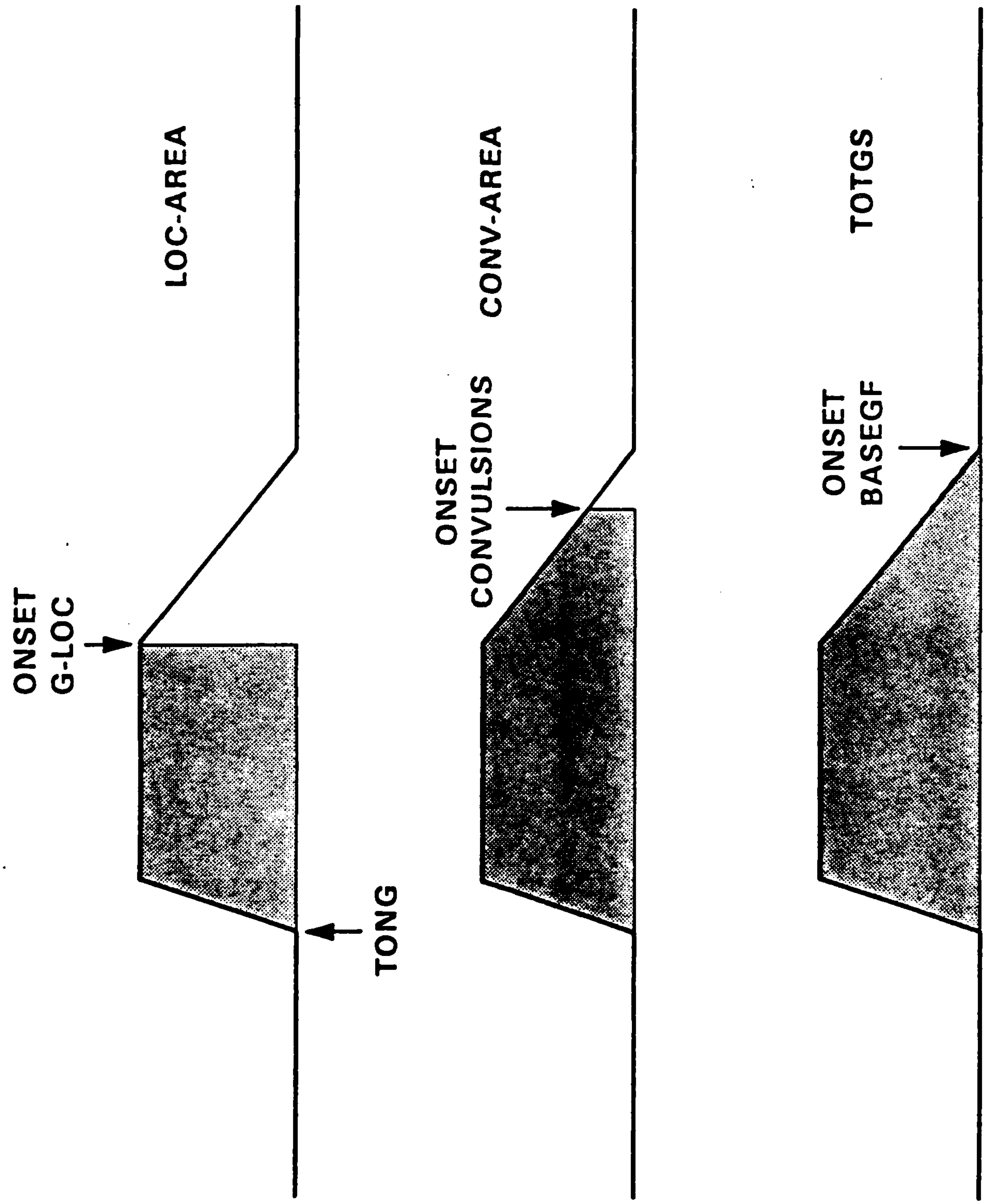

FIGURE 4. Area Under the $+G_z$-Time Exposure Curves.

Table 5. G-LOC Acceleration - Time Exposure Description.

PARAMETERS	UNITS	DESCRIPTION
TOTGS	G • s	The integrated area under the G • time exposure curve totally $\mathrm{TOTGS} = \frac{(\mathrm{GMAX}-1)\,(\mathrm{TGMAX}-\mathrm{TOG})}{2} + (\mathrm{GMAX}-1)\,(\mathrm{TOG}-\mathrm{TGMAX}) + \frac{(\mathrm{GMAX}-1)\,(\mathrm{TONBG}-\mathrm{TOG})}{2}$
CONV-AREA	G • s	The integrated area under the G • time exposure curve until the onset convulsions $\mathrm{CONV\text{-}AREA} = \frac{(\mathrm{GMAX}-1)\,(\mathrm{TGMAX}-\mathrm{TOG})}{2} + (\mathrm{GMAX}-1)\,(\mathrm{TOG}-\mathrm{TGMAX}) + \frac{(\mathrm{TONCON}-\mathrm{TOG})\,[(\mathrm{GMAX}-1) + (\mathrm{GCONV}-1)]}{2}$
LOC-AREA	G • s	The integrated area under the G • time exposure curve until the onset of unconsciousness $\mathrm{LOC\text{-}AREA} = \frac{(\mathrm{GMAX}-1)\,(\mathrm{TGMAX}-\mathrm{TOG})}{2} + (\mathrm{GMAX}-1)\,(\mathrm{TOG}-\mathrm{TGMAX})$

Mean, maximum, minimum, and standard deviation (±SD) were calculated for the G-LOC parameters. Analysis of variance and student's T-tests were used to compare the various data sets where appropriate.

RESULTS

The mean, maximum, minimum, and ±SD for the acceleration exposure, incapacitation, and myoclonic convulsion data for the entire 501 G-LOC episodes is given in Tables 6, 7, and 8. These tables summarize the characteristics of the entire G-LOC group. However, because of marked differences in the exposure conditions (G-onset rates, performance of an anti-G straining maneuver or remaining relaxed, and other differences) the data deserved further breakdown into more homogeneous subgroups. In spite of the differences in exposure conditions, very significant correlation between various parameters was found when considering the data for the entire study group as shown in Table 9. Onset rate and offset rate were significantly negatively correlated with G-LOC incapacitation, indicating that the slower the induction of G-LOC, and the slower the reduction of the $+G_z$-stress, the longer the incapacitation which results. This aspect of increased length of incapacitation was also observed with increased length of exposure to $+G_z$, as evidenced by the significant positive correlation of the total $+G_z$-exposure time, the total $+G_z$-seconds to onset of G-LOC and onset of convulsions, and the time to induce unconsciousness with G-LOC incapacitation times. The longer the exposure to $+G_z$, with it's prolonged reduction in CNS blood flow, the greater the physiologic insult with resulting increased time for subsequent recovery. To further evaluate the effect of onset rate on G-LOC incapacitation, the entire group was separated into subgroups based on the available ranges of $+G_z$-onset rates. Since the $+G_z$ exposures did not consist of a continuous spectrum of onset rates, arbitrary ranges based on the distribution frequency of available data were developed. Very gradual onset rates (VGOR) were considered as onsets between 0.060 - 0.083 G/s, gradual onset rates (GOR) were considered as onsets between 0.083 - 0.583 G/s, rapid onset rates (ROR) were considered as onsets between 0.60 - 1.75 G/s, intermediately high onset rates

Table 6. Acceleration Exposure Results for the Entire G-LOC Study Group.

VALUE*	BASEGO	ONRATE	GMAX	G-LOC	OFFRATE	BASEGF	TOTGS	LOCINDTI
MEAN	1.1	1.3	7.6	7.2	0.8	1.0	191	43.1
MAX	1.7	7.6	9.0	9.0	7.9	2.0	500	106.0
MIN	1.0	0.06	2.5	2.3	0.1	1.0	14	2.0
±SD	0.2	1.5	1.4	1.7	0.5	0.2	132	35.7
N	501	482	501	501	491	501	482	482

* See other tables for definition of terms. Mean, maximum (MAX), minimum (MIN), ±SD, and number of measurements (N) are given for each parameter.

Table 7. Incapacitation (seconds) Results for the Entire G-LOC Study Group.

VALUE*	ABSINCAP	RELINCAP-L	RELINCAP-S	TOTINCAP-L	TOTINCAP-S
MEAN	11.9	15.3	16.2	28.3	28.9
MAX	38.0	97.0	99.0	110.0	112.0
MIN	2.0	2.0	1.0	9.0	7.0
±SD	5.0	12.8	12.5	13.8	13.5
N	499	323	368	323	368

* See other tables for definition of terms. All incapacitation times are reported in seconds.

Table 8. Myoclonic Convulsions Results for the Entire G-LOC Study Group.

VALUE*	LOC-CONV	TOG-CONV	CONV-PER	CONV-ROC	GCONV
MEAN	7.7	7.5	3.9	1.1	2.3
MAX	20.0	23.0	10.0	22.0	9.2
MIN	-2.0	-6.0	1.0	-3.0	1.0
±SD	4.0	4.1	1.6	2.3	1.7
N	316	312	316	315	316

* See other tables for definition of terms. Kinetic description of myoclonic convulsions are given in seconds with the +Gz-level at onset of convulsions (GCONV) given in G-units.

Table 9. Correlation of Acceleration Exposure Parameters with Incapacitation for Entire G-LOC Study Group.

	ABSINCAP		TOTINCAP-L		TOTINCAP-S	
	CORR. COEF.	PROB.	CORR. COEF.	PROB.	CORR. COEF.	PROB.
ONRATE	-0.22	<0.0001	-0.31	<0.0001	-0.30	<0.0001
OFFRATE	-0.24	<0.0001	-0.11	<0.05	-0.11	<0.05
TOTGS	0.45	<0.0001	0.31	<0.0001	0.34	<0.0001
LOC-AREA	0.44	<0.0001	0.31	<0.0001	0.34	<0.0001
CONV-AREA	0.50	<0.0001	0.31	<0.0001	0.34	<0.0001
LOCINDTI	0.39	<0.0001	0.32	<0.0001	0.35	<0.0001

(IHOR) were considered as onsets between 1.90 - 2.67 G/s, and very high onset rates (VHOR) were considered as onsets between 2.80 - 7.60 G/s. Separation into these various onset ranges resulted in the incapacitation parameters shown in Table 10. The absolute incapacitation period showed considerable variation based on the separation of the data into onset range subgroups. The total incapacitation period was consistently longer for the more gradual onset rates. The most likely reason for the shorter absolute incapacitation for the VGOR subgroup is related to the extremely gradual onset allowing early recognition of the onset of unconsciousness and reduction of the +Gz-stress. Overall, this would serve to offset the prolonged decrease in central nervous system blood flow characteristic of the gradual onset exposure in general. In addition, the mean GMAX was less for the VGOR exposures (+6.3G_z) as compared to the GOR exposures (+7.7G_z), and the mean +G_z-offset rate for the VGOR exposures (0.84 G/s) was greater than for the GOR exposures (0.59 G/s). These two factors would also serve to reduce the physiologic insult for VGOR exposure as compared to GOR exposures. Since these kind of complicating factors were present, the remainder of the analyses were performed separating the data into only gradual onset (GOR) and rapid onset (ROR) data, GOR being less than 0.6 G/s, and ROR being greater than 0.6 G/s.

Considering all G-LOC episodes, 70% had associated myoclonic convulsive episodes. For the G-LOC episodes resulting from GOR exposures, 70% had associated myoclonic convulsive episodes, compared to 72% for ROR exposures. Table 11 compares the incapacitation resulting from G-LOC with and without myoclonic convulsions. Episodes of G-LOC with myoclonic convulsions result in longer incapacitation than episodes without myoclonic convulsions. Episodes of G-LOC with myoclonic convulsions occurring on GOR exposure have longer incapacitation than GOR exposure without myoclonic convulsions. The same was true for ROR exposure, myoclonic convulsions were associated with longer incapacitation. Episodes of G-LOC with myoclonic convulsions resulting from GOR exposure have longer incapacitation than ROR exposure with myoclonic convulsions. Finally, episodes of G-LOC

Table 10. The Effect of Variation of +Gz-Onset Rate on G-LOC Incapacitation.

MEAN PARAMETERS	+G_Z-ONSET RATE RANGES				
	VGOR	GOR	ROR	IHOR	VHOR
ONRATE (G/s)	0.07	0.13	1.16	2.39	3.69
OFFRATE (G/s)	0.84	0.59	0.81	1.05	1.00
TOTGS (G • s)	213	306	79	94	93
CONV-AREA (G • s)	192	259	53	63	62
LOC-AREA (G • s)	203	314	68	87	83
LOCINDTI (s)	73.7	72.0	11.1	9.3	7.3
TAG (s)	—	—	5.5	7.0	7.3
ABSINCAP (s)	10.7	14.3	9.3	11.2	10.7
TOTINCAP-L (s)	33.5	32.8	23.7	24.7	22.8
TOTINCAP-S (s)	32.4	34.4	20.3	26.4	23.7
N	69	188	64	54	107

Table 11. The Relationship of Incapacitation Associated with Onset Rate and Myoclonic Convulsions.

	INCAPACITATIONS (s)				
COMPARISON GROUPS	ABSINCAP	RELINCAP-L	RELINCAP-S	TOTINCAP-L	TOTINCAP-S
a. TOTAL GROUP					
(+) CONVULSIONS	12.7*	16.0	16.6	29.4°	29.9
(-) CONVULSIONS	9.9*	14.2	15.6	25.3°	26.6
b GOR EXPOSURES					
(+) CONVULSIONS	14.2+	18.4	19.5	34.1	34.9
(-) CONVULSIONS	11.2+	16.7	18.6	29.5	31.7
c. ROR EXPOSURES					
(+) CONVULSIONS	11.0*	13.3	13.7	24.4	24.8
(-) CONVULSIONS	8.4*	12.0	12.8	21.5	21.8
d. (+) CONVULSIONS					
GOR	14.2*	18.4-	19.5	34.1*	34.9*
ROR	11.0*	13.3-	13.7	24.4*	24.8*
e. (-) CONVULSIONS					
GOR	11.3-	16.7	18.6°	29.5-	31.7-
ROR	8.4-	12.0	12.8°	21.5-	21.8-

*p <0.0001

+p <0.001

-p <0.005

°p <0.05

without myoclonic convulsions resulting from GOR exposure have longer incapacitation than ROR exposure without myoclonic convulsions.

The episodes of G-LOC associated with wearing an anti-G suit were compared with the G-LOC episodes where an anti-G suit was not worn, as shown in Table 12. Anti-G suit wear was associated with an increased GMAX and GLOC. The ONRATE associated with anti-G suit wear was much more rapid (2.4 G/s) than the ONRATE where an anti-G suit was not worn (0.7 G/s). The incapacitation associated with G-LOC episodes where an anti-G suit was worn was decreased compared to the G-LOC episodes where the anti-G suit was not worn. This demonstrates a valuable, but previously unknown, facet of the protection provided by anti-G suits.

The episodes of G-LOC associated with the performance of an anti-G straining maneuver were compared with the G-LOC episodes where the subject was not performing the anti-G straining maneuver (relaxed) as shown in Table 13. Performance of the anti-G straining maneuver was associated with an increased GMAX and GLOC. The ONRATE was the same, however the LOCINDTI and TOTGS were much longer when the anti-G straining maneuver was performed. The incapacitation associated with G-LOC episodes where the anti-G straining maneuver was not performed (relaxed) was decreased as compared to the G-LOC episodes in which the maneuver was performed. The data does not provide an indication that the anti-G staining maneuver prolongs unconsciousness or incapacitation since the exposure characteristics were significantly different.

Although limited data was available relative to the presence or absence of a dream experience associated with the G-LOC episode, the data were analyzed to determine if dreams were associated with any unique G-LOC parameters, as shown in Table 14. The episodes of G-LOC which had associated

Table 12. The Effect of Wearing the Anti-G Suit on G-LOC Parameters.

	ANTI-G SUIT	
	(N=175)	(N=326)
PARAMETERS	YES	NO
ONRATE(G/s)	2.4(1.4)*	0.7(1.2)*
GMAX(G)	8.1(1.3)*	7.4(1.4)*
OFFRATE (G/s)	1.0(0.3)*	0.7(0.5)*
TAGS (s)	6.4(4.6)*	1.9(3.4)*
TOTGS (G • s)	99(49)*	239(136)*
GLOC (G)	7.9(1.4)*	6.8(1.7)*
LOCINDTI (s)	13.2(14.4)*	58.3(33.6)*
ABSINCAP (s)	10.3(3.5)*	12.8(5.5)*
RELINCAP-L (s)	13.9(12.6)	16.1(12.9)
RELINCAP-S (s)	14.8(12.3)	16.9(12.5)
TOTINCAP-L (s)	24.8(12.9)+	29.9(13.9)+
TOTINCAP-S (s)	25.6(12.5)+	30.4(13.7)+
GCONV (G)	3.3(1.8)	2.9(1.8)
LOC-CONV (s)	6.3(2.6)*	8.3(4.3)*
CONV-PER (s)	3.9(1.3)	3.9(1.7)
CONROC (s)	0.7(1.6)°	1.2(2.6)°

*$p<0.0001$

+$p<0.001$

°$p<0.05$

Table 13. The Effect of the Anti-G Straining Maneuver
on G-LOC Parameters.

	ANTI-G STRAINING MANEUVER	
	(N=449)	(N=51)
PARAMETERS	YES	NO
ONRATE (G/s)	1.3(1.6)	1.3(1.1)
GMAX(G)	7.8(1.3)*	5.9(1.3)*
OFFRATE (G/s)	0.8(0.5)	0.8(0.3)
TOTGS (G • s)	206(132)*	71(45)*
TAG (s)	3.2(4.5)*	5.0(2.6)*
GLOC (G)	7.4(1.6)*	5.7(1.5)*
LOCINDTI (s)	46.1(36.0)*	17.7(20.5)*
ABSINCAP (s)	12.2(5.1)+	10.3(3.8)+
RELINCAP-L (s)	15.7(12.9)°	8.3(7.9)°
RELINCAP-S (s)	17.1(12.6)*	6.1(4.0)*
TOTINCAP-L (s)	28.6(13.9)°	20.8(8.6)°
TOTINCAP-S (s)	30.0(13.5)*	16.8(5.0)*
GCONV (G)	3.1(1.8)*	2.2(1.1)*
LOC-CONV (s)	7.9(4.1)°	6.5(2.2)°
CONV-PER (s)	3.9(1.6)	4.0(1.6)
CONROC (s)	1.1(2.3)	0.7(2.2)

*$p<0.0001$

+$p<0.001$

°$p<0.005$

Table 14. Characteristics of G-LOC Acceleration Exposures
With and Without Dream Occurrences.

	DREAM OCCURRENCE	
	(N=87)	(N=16)
PARAMETERS	YES	NO
ONRATE (G/s)	1.1(1.4)	1.4(1.3)
GMAX(G)	7.6(1.4)	7.8(1.2)
GLOC (G)	7.2(1.5)	7.4(1.4)
OFFRATE (G/s)	0.7(0.3)	0.7(0.3)
TOTGS (G • s)	212(139)	172(118)
TAG (s)	2.6(3.5)	4.5(4.2)
LOCINDTI (s)	48.4(36.5)	35.1(33.3)
ABSINCAP (s)	14.2(5.6)°	10.6(3.7)°
RELINCAP-L (s)	18.0(13.9)	16.9(10.9)
RELINCAP-S (s)	18.4(13.4)	17.4(10.8)
TOTINCAP-L (s)	32.8(14.8)	28.1(11.8)
TOTINCAP-S (s)	33.0(14.2)	27.9(12.4)
GCONV (G)	2.6(1.7)*	3.3(1.0)*
TOG-CONV (G)	8.7(4.4)*	4.7(2.1)*
CONV-PER (G)	4.1(1.5)	3.8(0.8)
CONROC (G)	1.2(2.4)	2.1(2.6)

*p<0.0001

°p<0.002

dreams had increased incapacitation, TOG-CONV, CONV-PRN, and decreased GCONV. Dreams were therefore associated with exposure characteristics producing a more severe CNS insult.

Certain G-LOC episodes had intervention during the recovery period. The intervention inadvertently came from the centrifuge central observer. It usually resulted from concern for the subject following return of consciousness. The central observer interaction consisted of verbal prompting or questioning of the subject prior to his spontaneously responding to the warning light/sound. It was of interest to evaluate the exposure and response parameters associated with the G-LOC episodes in which central observer interaction (COI) occurred as shown in Table 15. The incapacitation for the COI episodes was increased, essentially because of the increased relative incapacitation. There was little indication that anything unique caused these subjects to be less responsive to the light/sound warning. Although these subjects may have suffered increased confusion and disorientation, the minimally increased absolute incapacitation and similar exposure characteristics did not provide insight into a physiologic cause. Myoclonic convulsions occurred for 65% of the G-LOC episodes which required COI and for 69% of the G-LOC episodes without COI.

To further evaluate the G-LOC episodes with myoclonic convulsions, the episodes were separated into the episodes with the shortest convulsions (1s), N = 11, and the longest convulsions (8-10s), N = 8. Since the myoclonic convulsions were very tightly grouped around the mean 4s period, few episodes were either extremely short or extremely long. Only these small groups were available for analysis. The G-LOC episodes with long convulsions were characterized by increased absolute incapacitation and total incapacitation, and more gradual onset rates as shown in Table 16.

The incapacitation resulting from G-LOC was further evaluated by separating the data into two extremes based on the upper 10% and lower 10% of the entire group based on absolute, relative, and total in-

Table 15. The Effect of Central Observer Interaction on Recovery from G-LOC.

PARAMETERS	Central Observer Interaction (N=57) YES	(N=319) NO
ONRATE (G/s)	1.2(1.5)	1.6(1.6)
GMAX(G)	8.0(1.0)	8.1(1.1)
OFFRATE (G/s)	0.9(1.1)	0.7(0.3)
GLOC (G)	7.7(1.2)	7.7(1.3)
TAG (s)	3.0(4.5)	3.7(4.0)
TOTGS (G • s)	227(134)	202(136)
LOCINDTI (s)	49.3(35.2)	41.8(36.4)
ABSINCAP (s)	13.8(5.3)	12.5(5.0)
RELINCAP-L (s)	33.5(17.5)*	11.9(8.1)*
RELINCAP-S (s)	35.5(17.3)*	12.7(7.1)*
TOTINCAP-L (s)	47.5(17.6)*	24.7(9.3)*
TOTINCAP-S (s)	49.4(17.0)*	25.2(8.7)*
GCONV (G)	3.2(1.8)	3.3(1.8)
LOC-CONV (s)	9.6(4.8)°	8.0(3.9)°
CONV-PER (s)	3.6(1.6)	3.8(1.5)
CONROC (s)	1.6(2.2)	1.2(2.6)

*p0.0001

°p0.05

Table 16. The Characteristics of G-LOC Episodes Associated with the Length of Myoclonic Convulsions.

	MAX-MIN CONVULSIONS	
	(1s)	(8-10s)
PARAMETERS	MIN CONV	MAX CONV
ONRATE (G/s)	2.0	0.4
GMAX(G)	7.0	7.4
GLOC (G)	6.9	6.9
OFFRATE (G/s)	0.8	0.6
ABSINCAP (s)	11.7	20.0
RELINCAP-L (s)	16.7	8.5
RELINCAP-S (s)	17.0	11.3
TOTINCAP-L (s)	28.5	31.8
TOTINCAP-S (s)	28.7	34.7
BASEGF (G)	1.1	1.0
N	11	8

capacitation. The characteristic parameters associated with the extremes were then calculated as shown in Tables 17, 18, and 19. The longest absolute incapacitation was associated with a higher GMAX, a slower onset rate, a longer $+G_z$-exposure, and a higher $+G_z$-level at onset of G-LOC. The myoclonic convulsions lasted slightly longer, occurred at a time more prolonged from the onset of G-LOC, and required a longer time from the end of convulsions to the return of unconsciousness. When absolute incapacitation was long, relative, and therefore total, incapacitation was also prolonged. The longest total incapacitation was associated with many of the same characteristics of the longest absolute incapacitation. The characteristics of the longest relative incapacitation were less differentiated from the shorter relative incapacitation, probably indicating that relative incapacitation is less dependent on strict physiologic determinants.

DISCUSSION

Previous reports have qualitatively described G-LOC and associated phenomenon (2,3). To date, no thorough quantitative or kinetic description has been given, even though centrifuge and inflight G-LOC has been observed for over 60 years (8). The methods used to define G-LOC in this report not only describe the events that occur during G-LOC, but also establish the kinetic relationship between these events. G-LOC results from exceeding the body's tolerance to head-to-foot acceleration stress. Unconsciousness results from inadequate blood flow to the central nervous system (CNS). The period of unconsciousness has been defined as the absolute incapacitation period, since a period of relative incapacitation, characterized by confusion and disorientation, follows unconsciousness. The sum of the unconsciousness and the confusion and disorientation is defined as the total incapacitation period which represents the period of time a pilot would not be in control of his fighter aircraft. This relationship, as shown in Figure 5, is extremely important for understanding G-LOC as it pertains to pilot safety. In addition to unconsciousness and subsequent incapacitation, myoclonic convulsions result when the is-

Table 17. Characteristics of the Upper and Lower Extremes of the Absolute Incapacitation Period.

PARAMETERS	ABSOLUTE INCAPACITATION UPPER 10%	LOWER 10%
GBASE (G)	1.0	1.1
ONRATE (G/s)	0.3	1.2
GMAX(G)	8.1	7.0
GBASEF (G)	1.0	1.0
OFFRATE (G/s)	0.5	0.9
TAG (s)	0.6	3.8
TOTGS (G • s)	323	127
GLOC (G)	7.9	6.3
OFF-AREA-L (G • s)	50	30
OFF-AREA-C (G • s)	45	7
LOCINDTI (s)	74	32
ABSINCAP (s)	23.1	4.2
RELINCAP-L (s)	13.4	11.3
RELINCAP-S (s)	14.5	13.8
TOTINCAP-L (s)	36.1	15.7
TOTINCAP-S (s)	37.4	18.1
GCONV (G)	1.9	1.3
LOC-CONV (s)	13.8	2.0
CONV-PER (s)	5.0	3.0
CONROC (s)	3.9	0.5

Table 18. Characteristics of the Upper and Lower Extremes of the Total Incapacitation Period.

PARAMETERS	TOTAL INCAPACITATION UPPER 10%	TOTAL INCAPACITATION LOWER 10%
GBASE (G)	1.1	1.2
ONRATE (G/s)	0.8	2.5
GMAX(G)	7.9	7.8
GBASEF (G)	1.0	1.1
OFFRATE (G/s)	0.8	1.0
TAG (s)	3.2	6.6
TOTGS (G • s)	246	90
GLOC (G)	7.6	7.5
OFF-AREA-L (G • s)	41	25
OFF-AREA-C (G • s)	35	20
LOCINDTI (s)	54	12
ABSINCAP (s)	14.6	8.0
RELINCAP-L (s)	41.8	4.2
RELINCAP-S (s)	45.9	4.8
TOTINCAP-L (s)	56.0	12.3
TOTINCAP-S (s)	60.6	12.9
GCONV (G)	2.5	2.2
LOC-CONV (s)	9.8	4.9
CONV-PER (s)	3.3	3.4
CONROC (s)	1.2	0.1

Table 19. Characteristics of the Upper and Lower Extremes of the Relative Incapacitation Period.

PARAMETERS	RELATIVE INCAPACITATION UPPER 10%	LOWER 10%
GBASE (G)	1.1	1.3
ONRATE (G/s)	1.1	2.5
GMAX(G)	7.8	8.0
GBASEF (G)	1.0	1.2
OFFRATE (G/s)	0.9	1.0
TAG (s)	3.4	6.5
TOTGS (G • s)	211	118
GLOC (G)	7.4	7.9
OFF-AREA-L (G • s)	37	28
OFF-AREA-C (G • s)	32	25
LOCINDTI (s)	47	19
ABSINCAP (s)	13.0	14.1
RELINCAP-L (s)	40.8	3.1
RELINCAP-S (s)	46.3	2.6
TOTINCAP-L (s)	53.8	19.5
TOTINCAP-S (s)	59.2	16.8
GCONV (G)	2.4	2.1
LOC-CONV (s)	9.3	8.0
CONV-PER (s)	3.4	5.1
CONROC (s)	1.2	2.7

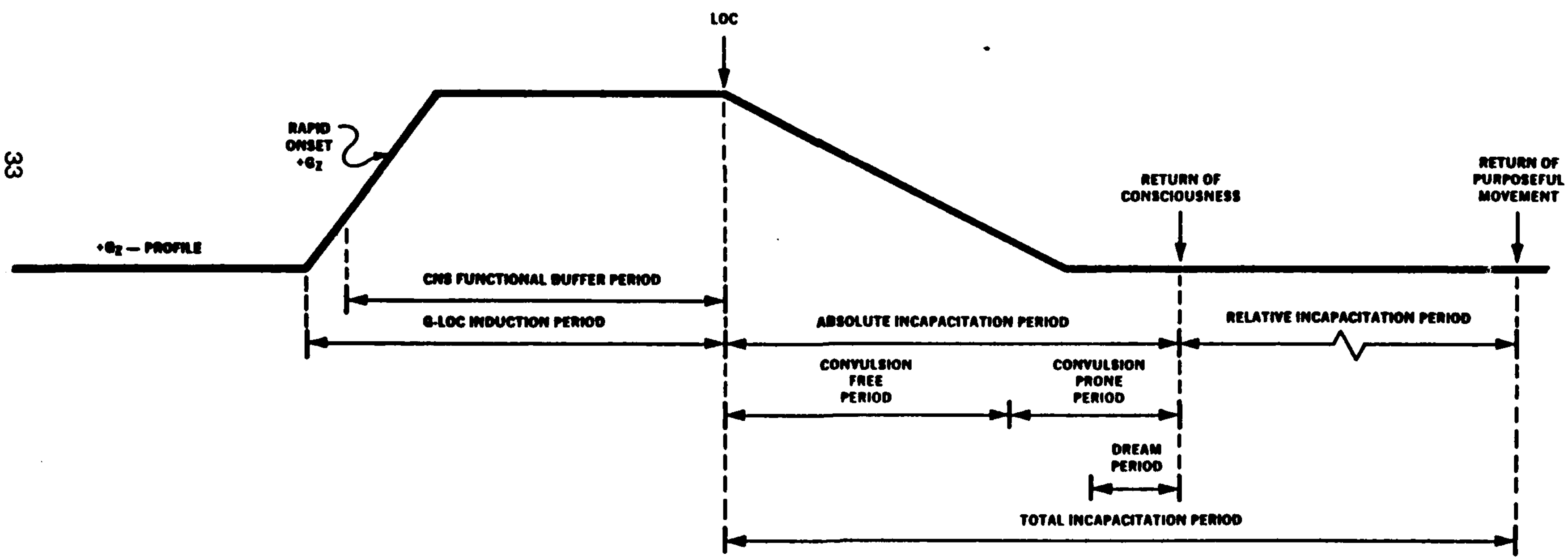

FIGURE 5. $+G_Z$-Induced Loss of Consciousness Psychophysiologic Description.

chemic/hypoxic insult to the CNS is sufficiently great. These myoclonic convulsions occur in a specific relationship to the unconsciousness and recovery. The myoclonic convulsions occur after onset of unconsciousness and follow the return of CNS blood flow, as determined by temporal artery doppler flow measurements. Based on when myoclonic convulsions occur, the absolute incapacitation period can be divided into an initial convulsion free period followed by a convulsion prone period as shown in Figure 5. The myoclonic convulsions consistently last approximately 4 seconds, and end virtually coincident with the return of consciousness. Dreams were also documented to occur in association with the G-LOC episode. The physiologic mechanisms which are responsible for memorable dreams may be of importance in establishing the psychophysiologic basis of G-LOC and subsequent recovery. It has been postulated that dreams occur near the end of the absolute incapacitation and convulsion prone periods as shown in Figure 5. This has been suggested based on the seemingly frequent incorporation of physiologic events (myoclonic convulsive movements and a sense of paralysis and helplessness) into many of the reported dreams. This would indicate that as CNS blood flow returns, the functional capabilities of the CNS return in a sequential manner. The CNS circuitry for dreaming and memory returns to a functional state prior to the return of full consciousness.

A thorough review of the literature relating to unconsciousness is difficult since investigation in this area is reported in a diverse manner. Unconsciousness data is reported as fainting, syncope, and as acute anoxia, hypoxia, or ischemia. Frequently, important information is also included in reports on fits, seizures, strokes, and acute cardiac alterations (sudden death). Engel's 1950 book on fainting remains current and is a classic (1). Although an extremely interesting and important physiologic phenomenon, clinical research does not include unconsciousness as a primary medical problem. It is considered a symptom of an underlying medical (or physiological) problem. Aerospace medicine, on the other hand, has to deal with completely healthy aircrew, with G-LOC being a primary problem for aircrew of fighter type aircraft.

The detailed kinetic sequence of G-LOC and associated events required measurement and calculation of the parameters defined in Tables 1 through 5 and Figures 1 through 4. A formal description of unconsciousness and techniques to kinetically quantify it has not previously been developed. Previous investigations of G-LOC and other forms of unconsciousness have described the characteristics of unconsciousness, including the occurrence of convulsive activity. Few, if any, of these investigations have had the opportunity to have permanent video recording of a large number of loss of consciousness episodes for detailed analysis.

The major aspects of importance when considering the entire group of G-LOC episodes include the kinetics of resulting incapacitation and the myoclonic convulsions. The mean length of unconsciousness (absolute incapacitation) resulting from G-LOC was 11.9 (±5.0) seconds. The resulting confusion and disorientation (relative incapacitation) extends the incapacitation an additional 15.3 (±12.8) seconds, as determined by a purposeful movement to disengage the warning light and 16.2 (±12.5) seconds to disengage the auditory warning. The total incapacitation resulting from G-LOC, which might be considered the length of time a fighter pilot would not be in control of his aircraft, should G-LOC occur, was 28.3 (±13.8) seconds, as measured by warning light disengagement, and 28.9 (±13.5) seconds, as measured by auditory warning disengagement. The ranges of the unconscious period and the total incapacitation are also important, especially the maximum values, which have the greatest potential for resulting in aircraft mishaps. The absolute incapacitation ranged from a minimum of 2 seconds to a maximum of 38 seconds, and the total incapacitation (as measured by warning light response) ranged from a minimum of 9 seconds to a maximum of 110 seconds. The absolute incapacitation period is made up of a convulsion free period beginning immediately upon onset of G-LOC and lasting on the average 7.7 seconds. This is followed by a convulsion prone period which lasts on the average 3.9 seconds. The myoclonic convulsions end almost simultaneously with the return of consciousness. The difference between the end of myoclonic convulsions and the return of consciousness (CON-ROC) is given as 1.1 seconds.

This differential is, in fact, even less than this value, since the measurements were made only to the nearest 1 second. Mechanistically, the sequence was almost exclusively measurement of the end of convulsions followed instantly by the return of consciousness. It was clearly evident after observing the entire group of individuals who had myoclonic convulsions, that such convulsions end virtually coincident with the return of consciousness As can be seen, the length of myoclonic convulsions remained very consistently fixed at approximately 4.0 seconds, irrespective of how the data was separated or subgrouped.

It was very clear that the incapacitation resulting from G-LOC was highly dependent on the type of $+G_z$-exposure. As shown in Table 9, incapacitation was negatively correlated with $+G_z$-onset and offset rates. Longer incapacitation resulted from slower (more gradual) onset rate profiles, which by experimental design have slower offset rates. The longer the G-LOC induction period (LOCINDTI), which is also associated with gradual onset exposures, the longer the incapacitation periods. As would have been suspected, the greater the area under the $+G_z$-time exposure curve (for the total G • s exposure, the G • s area to unconsciousness, and the G • s area to the onset of myoclonic convulsions), the longer the resulting incapacitation. A more complex picture arises upon relating the various onset rate subgroups as shown in Table 10. Very gradual onset $+G_z$-stress, for instance, had a slower onset rate, a higher offset rate, a smaller area under the $+G_z$-time exposure curve, and almost identical loss of consciousness induction times compared to gradual onset $+G_z$-stress. This complex situation prevents an exact relationship of very narrow onset rate ranges with incapacitation.

There is little doubt that the more gradual onset exposure results in increased incapacitation as compared to rapid onset exposure. The gradual onset exposure therefore must result in a greater central nervous system insult than rapid onset exposure. The prolonged period of reduction in central nervous system blood flow results in a longer period of unconsciousness and time required to gain recovery.

This type of relationship would have been predicted; however, it has not been previously physiologically demonstrated. The absolute incapacitation period (and therefore total incapacitation) would appear to be determined by the magnitude of the CNS physiologic insult (length, location, and amount of ischemia/hypoxia). The relative incapacitation, although definitely proportional to the magnitude of the physiologic insult, may also be related to other factors. The ability to reorient and respond to the warning light and auditory tone is more likely to depend on such factors as previous G-LOC experience and motivation. Previous results have indicated that "G-LOC training" may result in significantly decreasing the relative incapacitation period (9).

Convulsive activity has been frequently associated with unconsciousness induced by various techniques. Reports have indicated that convulsive activity occurs in approximately 11.9% of blood donors who have syncopal episodes (10). Franks reported that convulsive activity occurred in 52% of centrifuge G-LOC episodes (2). The myoclonic convulsions which occur during G-LOC may occur more frequently than occurs in routine syncope, since the individual is restrained in the upright sitting position during $+G_z$-stress. The finite period required for offset of the $+G_z$-stress is also very likely prolonged over that experienced when the common faint results in falling to a horizontal position. As an example, the time to return to $+1.0G_z$ from $+8.0G_z$ at 0.8 G/s requires 8.8 seconds. Myoclonic convulsions occur with exposures that produce longer incapacitation. Exposures with a greater CNS insult not only prolong incapacitation but are more frequently associated with myoclonic convulsions. Clonic, tonic, and tonic-clonic convulsive activity has been described in a number of studies involved with unconsciousness. The myoclonic convulsions induced during G-LOC occur following return of blood flow to the CNS. This would suggest they are a reperfusion phenomenon. Prolonged reduction of CNS blood flow also produces convulsions; however descriptions indicate that with prolonged ischemia tonic convulsions are more frequent and are preceded by clonic-type activity (if it occurs).

To gain further insight into the characteristics of G-LOC related myoclonic convulsions, the extremes of the duration of myoclonic convulsions were evaluated as shown in Table 16. The minimum length convulsions (1s) were associated with rapid onset, rapid offset, and decreased absolute and total incapacitation. The fact that relative incapacitation is not uniformly predicted by absolute incapacitation once again suggests that it is less dependent on purely physiologic determinants. The maximum length convulsions (8-l0s) were associated with gradual onset, slower offset, and longer absolute and total incapacitation.

The effect of wearing an anti-G suit on the G-LOC parameters is of significant importance to aerospace physiology. As shown in Table 12 anti-G suit wear as associated with rapid onset to higher $+G_z$-levels. G-LOC occurred at significantly higher $+G_z$-levels as would be expected. Very importantly, the anti-G suit was associated with significantly shortened absolute and total incapacitation. This may be a reflection of the type of exposure (rapid onset, which by itself also has shorted incapacitation). However, the higher $+G_z$-*levels would have at least partially countered* the isolated effect of onset rate. The onset of myoclonic convulsions following onset of G-LOC occurred earlier when an anti -G suit was worn. This may be a result of a more rapid return of CNS blood secondary to the more rapid offset of the $+G_z$-stress, the wear of the anti-G suit, or a combination of the two. The duration of the myoclonic convulsions were of exactly the same duration. Although of very slight (but statistically significant) magnitude, non-wear of the anti-G suit was found to have a prolonged time from the termination of myoclonic convulsions to the return of consciousness. This would seem to be most likely resultant from the prolonged and slower rate of return of CNS blood flow.

The performance or nonperformance of an anti-G straining maneuver was associated with similar onset and offset rate exposures, however performing the anti-G straining maneuver resulted in achieving a much higher $+G_z$-level along with G-LOC also occurring at a much higher $+G_z$-level. The duration (total G • s) of exposure was also considerably lengthened by performing the anti-G straining maneuver. All in-

capacitation parameters were prolonged with performance of the anti-G straining maneuver (to higher $+G_z$-levels). The myoclonic convulsions occurred at a higher $+G_z$-level, and occurred significantly later following G-LOC.

The data on the presence or absence of dream occurrence was limited because of the lack of accurate questioning of the subjects during the early years of G-LOC documentation. Although limited, the data was analyzed based on the presence or absence of dreaming. The occurrence of dreams was documented more frequently than non-occurrence of dreams simply because in the early episodes documentation occurred only when the subject offered up its occurrence. The exact frequency of dream occurrence associated with G-LOC is unknown. The types of $+G_z$-exposures were similar when comparing the presence or absence of dreams; however when dreams did occur, the absolute incapacitation was significantly longer, the $+G_z$-level at which the myoclonic convulsions occurred was higher, and the length of time from offset of $+G_z$ to the onset of myoclonic convulsions prolonged. The occurrence of dreams may result from a greater CNS insult which prolongs the absolute incapacitation. The characteristics of the dream content and possible physiologic mechanism have been described elsewhere (11). It was suggested that the actual dream period may be near the termination of both the absolute incapacitation period and the convulsion prone period. The presence of memorable dreams are of extreme importance relative to the non-invasive establishment of the physiologic mechanisms of G-LOC induction and recovery. The isolation of these vivid "dreamlets" which occur over such a short period of time may also be of specific interest to dream researchers. The fact that the dreams were memorable indicates that the mnestic CNS circuits become functional prior to return of "full" consciousness.

The effect of central observer interaction with the subject, which may have been expected to reduce the relative and total incapacitation, was found to be associated with the longer relative and total incapacitation. It is therefore evident that this interaction occurred most frequently when the subject recovering

from unconsciousness required a long time to react to the warning light/auditory tone. The slightly longer absolute incapacitation, which occurred during G-LOC episodes requiring central observer interaction, was not significant. A slightly longer time from the onset of unconsciousness to the onset of myoclonic convulsions did reach significance. It remains unknown whether or not voice interaction with a subject recovering from unconsciousness has an effect on the relative (and total) incapacitation.

A final subgrouping of the data was made to determine the characteristics of the parameters associated with the longest (upper 10%) and shortest (lower 10%) *incapacitation* as shown in Table 17. Once again the longest absolute incapacitation was associated with gradual onset, slower offset type exposures to higher $+G_z$-levels in which G-LOC occurred at a higher level. The convulsion free period (LOC-CONV) was increased, as was the duration of the myoclonic convulsions and the delay between the end of convulsions and the return of consciousness. This separation of the data graphically illustrates the type of exposure which results in the greatest insult to the CNS. Similar findings for the longest and shortest relative and total incapacitation were found as shown in Tables 18 and 19. The direct relation between the magnitude of CNS insult and the time required for recovery lends evidence to a mechanism for G-LOC that is not an all or nothing phenomenon.

The kinetic observations obtained in this investigation do provide a clearer understanding of what actually occurs during loss of consciousness. A method for experimental documentation of G-LOC and a framework for describing the G-LOC phenomenon has been established along with the results. Although the exact physiologic mechanism of unconsciousness in humans still remains to be determined, these results do establish a basis for formulating a physiologic mechanism of acceleration-induced loss of consciousness. Based on the predicted mechanical effects of $+G_z$-stress on reduced CNS blood flow, there would be a sequential reduction beginning at the top of the CNS and progressing downward. Unconsciousness results when the blood flow to the critical CNS area(s) responsible for maintaining conscious-

ness is reduced to a critical level for a specific length of time. Based on the most rapid onset $+G_z$-exposures it would seem apparent that the CNS tolerance to very abrupt reduction of blood flow is approximately 7s. The period of time the CNS can function without adequate blood flow has been termed the CNS functional buffer period. This CNS functional buffer period is slightly less than the G-LOC induction period since a certain level of $+Gz$ must be reached before reduction of CNS blood flow begins. This approximate 7s period required to induce G-LOC is very close to the 6.8s period previously measured to result in unconsciousness using acute cervical neck pressure in humans (12). As opposed to the almost instantaneous return of CNS blood flow in the cervical neck pressure studies, reduction of the $+G_z$-stress requires a longer period of time. Depending on the $+Gz$-level at which G-LOC occurs and the offset rate, there is a period of time in which CNS blood flow remains compromised after onset of G-LOC. Based on temporal artery doppler flow measurements, the total period of reduced flow characteristic of rapid onset $+G_z$-exposures is 12-14s. This CNS insult results in approximately 12s of unconsciousness (absolute incapacitation), and, at least 70% of the time, 4s of myoclonic convulsions which terminate virtually coincident with the return of consciousness. Although their frequency is unknown, memorable dreams are thought to occur near the end of the myoclonic convulsions, just prior to the return of consciousness. Based on this sequence of events, and previous research on convulsive activity (13), it may be postulated that high, sustained $+G_z$-stress results in a non-functional cerebral cortex (and thalamocortical tract system) which causes unconsciousness. The myoclonic convulsions may result from a disinhibited brainstem caudal reticular formation (the reticular formation is less affected by $+G_z$-stress because of its lower position in the $+G_z$ field and perhaps its greater resistance to ischemia/hypoxia). This would explain the coincident end of myoclonic convulsions and return of consciousness, as the cerebral cortex regains function and re-inhibits the reticular formation. Dream events may occur in the short period prior to return of consciousness as the structures necessary for dream production and memory return, ending as cortical function returns which re-inhibits these struc-

tures which were momentarily liberated from cortical control. A more complete description of this postulated mechanism has been given (14).

Incapacitation continues after return of consciousness in the form of confusion and disorientation. This period has been termed relative incapacitation which lasts approximately 12s with rapid onset $+G_z$-stress. The total incapacitation for this operational rapid onset $+G_z$-stress is approximately 24s, a very long period indeed for a fighter pilot not to be in control of his multi-million dollar aircraft should G-LOC occur. Psychologic alterations result from the G-LOC episodes as previously described (15). They are characteristic, in a mini-way, of what is observed in more severe and prolonged alterations of CNS blood flow. This includes decerebrate posturing, amnesic phenomenon, and transient psychologic phenomenon. Complete psychophysiologic recovery requires as long as 24 hours, earlier if a period of sleep allows a resetting of the fragmented thought processes that exist following G-LOC. It is evident that periods of altered performance persist longer than measured in this study. These alterations are of extreme importance to aircrew combat flight ability and must undergo further evaluation.

A final solution for fighter aviation related G-LOC requires a thorough knowledge and understanding of this problem. The kinetic aspects of unconsciousness in this large number of healthy humans, as thoroughly documented with videotape recording, should also be of interest to neuro-psychophysiologic researchers investigating unconsciousness, convulsive activity, memory, and dream phenomenon.

REFERENCES

1. Engel G.L. Fainting, 1950; Charles G. Thomas, Springfield, Illinois.

2. Franks W.R., Kerr W.K., Rose B. Some neurologic signs and symptoms produced by centrifugal force in man, J. Physiol. 1945; 104:10P-11P.

3. Lambert E.H., Wood E.H. The problem of blackout and unconsciousness in aviators, Med. Clin. N. Am. 1946; 30:833-844.

4. Whinnery J.E., Slaughter J.W. A computerized repository system for collection and retrieval of acceleration stress research data, Meth. Inform. Med. 1981; 20:10-15.

5. Whinnery J.E., Burton R.R., Boll P.A., Eddy D.R., Characterization of the resulting incapacitation following unexpected $+G_z$-induced loss of consciousness, Aviat. Space Environ. Med. 1987; 58:631-636.

6. Whinnery J.E., Methods for describing and quantifying $+G_z$-induced loss of consciousness, Aviat. Space Environ. Med. (submitted for publication).

7. Leverett S.D., Burton R.R., Physiologic effects of high sustained $+G_z$ force on man, (COSPAR) Life Sciences and Space Research, Vol XVII, R. Holmquest Ed., Pergamon Press Oxford 1979, 171-185.

8. Burton R.R., G-induced loss of consciousness: definition, history, current status, Aviat. Space Environ. Med. 1988; 59:2-5.

9. Whinnery J.E., Burton R.R., $+G_z$-induced loss of consciousness: a case for training exposure to unconsciousness, Aviat. Space Environ. Med. 1987; 58:468-472.

10. Lin J.T.Y., Ziegler D.K., Lai C.U., Bayer W., Convulsive syncope in blood donors, Ann. Neurol. 1982: 11:525-528.

11. Forster E.M., Whinnery J.E., Recovery from $+G_z$-induced loss of consciousness: psychophysiologic considerations, Aviat. Space Environ.Med. (in press).

12. Rossen R., Kabat R., Anderson J.P., Acute arrest of the cerebral circulation in man, Arch. Neurol. Psychiat. 1943; 50:510-528.

13. Gastaut H. , Fischer-Williams M., The physiopathology of epileptic seizures, Handbook of Physiology, Neurophysiology, Sect 1. Vol 1, Chapt. XIV, p. 329-363. American Physiologic Society; Washington, D.C. 1959.

14. Whinnery J.E., On the theory of $+G_z$-induced loss of consciousness, Aviat. Space Environ. Med. (submitted for publication).

15. Whinnery J.E., Jones D.R., Recurrent $+G_z$-induced loss of consciousness, Aviat. Space Environ. Med. 1987; 58:943-947.

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